MICHIGAN ENVIRONMENTAL SCIENCE BOARD

AIR PANEL
MEETING SUMMARY
WEDNESDAY, MARCH 26, 1997
PARK INN INTERNATIONAL
HOWELL, MICHIGAN

PANEL MEMBERS PRESENT
Dr. Raymond Demers
Dr. George Wolff
Dr. Ralph Kummler
Dr. Jack Harkema
Mr. Keith Harrison, MESB Executive Director & Chair pro tem

DMB/EAD SUPPORT STAFF PRESENT
Mr. Jesse Harrold, Environmental Officer

I. CALL TO ORDER

Mr. Harrison called the meeting of the Michigan Environmental Science Board (MESB) Air Quality Panel to Order at 9:00 AM.

II. EXECUTIVE DIRECTOR'S REPORT

Mr. Harrison announced that Dr. Fischer would not be able to attend the meeting. He also indicated that Dr. Kenneth Rosenman, Michigan State University (MSU) Medical School, has been added to the Air Quality Panel. He introduced Evelyn Thomas, a MSU graduate student, who will be working under a contract with the MESB, to research particulate matter (PM) and ozone epidemiological investigations under Dr. Rosenman's direction. Also, at the request of Dr. Demers, Dr. Sverre Vedal from the University of British Columbia, has agreed to act as adjunct consultant for the Panel, reviewing the Panel's drafts and final response to the Governor.

Mr. Harrison indicated that there is also a new MESB member, Dr. John A. Gracki, a chemist from Grand Valley State College, who is replacing Dr. Richard Cook. Dr. Cook took a position in Pennsylvania. Finally, Mr. Harrison indicated that on March 14, 1997, Executive Order 1997-3 was signed by the Governor transferring the MESB and the Environmental Administration Division from the Department of Management and Budget to the Department of Environmental Quality. In terms of MESB, the move will not cause any change except for, eventually, a different physical location of the office and different telephone and fax numbers. The Executive Order will become effective May 14, 1997.

III. PUBLIC COMMENT

Dennis Leonard, Detroit Edison, distributed copies of the U.S. Environmental Protection Agency's (USEPA) preamble to the proposed PM rule, highlighting the discussion of the variability of $PM_{2.5}$ to PM_{10} ratios among cities. In western cities during the winter months a formation of ammonium nitrates accounts for most $PM_{2.5}$. In the eastern U.S. ammonium nitrates are not substantial, but sulfates are; in northern communities, wood smoke accounts for much of the $PM_{2.5}$. The Electric Power Research Institute (EPRI), in comments to a U.S. Senate committee, stated that a new standard, like $PM_{2.5}$, requires dividing the substance to be controlled. The initial research results by EPRI, using samplers that work on the same principles as the USEPA reference methods, indicated that in some cities the reference method would not capture a substantial portion of fine particle constituents. Neither the EPRI nor the USEPA results has been published yet.

Mr. Jim Armelagos, public interest scientist, commented that measurable health effects have been tied to specific combustion compositions of atmospheric aerosols and mixtures of associated pollutants by Michael T. Kleiman of the University of California - Irvine. Research also suggests that long term exposure to the atmospheric aerosols induced by combined contaminants may have a larger effect on human health than acute short term exposures. The atmosphere distributes substances with complex toxic and carcinogenic potential. According to Mr. Armelagos, an environmental chemist at the Louisiana State University has shown that oxides of nitrogen merge with polycyclic aromatic hydrocarbons (PAHs) to form free radical reactions, making one of the more mutagenic, thus carcinogenic, pollutants, in urban air. There is an article in the July 1996 issue of *Environmental Health Perspectives* entitled “Dioxin Light Properties of Trichloroethylene Combustion Generated Aerosols”, in which the authors report the presence in particulate matter of at least 250 chlorinated incomplete combustion by-products, but not the obvious target compounds of tetrachlorodibenzo-p-dioxin and tetrachlorodioxin furans at detectable levels, suggesting that rare toxic effects may arise from substances other than those targeted by conventional chemical analyses. Mr. Armelagos stated that it is critical to have accurate chemical composition analyses of atmospheric aerosol samples taken from point sources. It would also be possible to determine the free radical or hydroxyl activity associated with $PM_{2.5}$ to estimate the ability of atmospheric aerosols to induce DNA damage by the generation of highly reactive species in the body. He disagreed with a March 21,1997 *Detroit News* article, which indicated that there was no agreed upon methodology for monitoring small particles. There is indeed available and effective measurement technology. Mechanical collectors, such as cascade impactors, and others, such as light scattering otometers and board diffusion battery analysis are commercially available. Source monitoring can be easily done with current sampling instrumentation.

Dr. Larry Holcomb, Holcomb Environmental Services, provided the Panel with some data on indoor/outdoor ratios and cautioned that if the new standard is adopted it will also impact indoor air quality standards, costing billions of dollars. He also said that none of the animal study data has thus far shown carcinogenic effects or biological

plausibility for the effects being suggested in the mortality studies. If there is increased mortality, it is an acute effect, and the available data do not show that it is $PM_{2.5}$. It also has not been established that other chemicals are not involved.

IV. PRESENTATION

Mr. Harrison introduced Dr. Joel Schwartz, Harvard School of Public Health, who spoke on available epidemiological investigations which have served as the basis for the National Resources Defense Council report and the USEPA proposed regulations on PM. A synopsis of his presentation may be found in Attachment 1.

Mr. Harrison asked Dr. Schwartz's opinion on the negative Abbey study. Dr. Schwartz replied that while David Abbey was still not saying exactly the same thing as everyone else, his conclusions, regarding seeing an association with all cause mortality and particulate air pollution exposure, were converging with other investigators. He also has a paper in review that shows an association between particulate air pollution exposure, and long-term lung function detriments.

Dr. Kummler asked if anyone had correlated the measured air pollution exposure with the actual exposures of individuals who were hospitalized due to air pollution. Dr. Schwartz indicated that no one has done so directly. Rather, the findings come from a series of statistical inferences regarding correlations between monitor to monitor measurements within the study area, $PM_{2.5}$ composition within PM_{10}, and indoor and outdoor air exposures.

Dr. Harkema asked what types of studies would be useful to help clarify the outstanding PM issues. Dr. Schwartz replied that first of all, the studies that have been done to date suggest to him that there is a biological plausibility, since animals with biologically relevant diseases can be killed at currently allowable particle concentrations. Beyond that, it would be useful to clarify why transition metals act the same in biological mortality, even though they may come from different sources. Also, additional studies on other cities with high pollution records, such as Los Angeles, could help clarify some of the questions brought up in the earlier studies. Finally, a study to determine the predisposition of the persons and animals to pulmonary hypertension and arrhythmia would be useful in targeting of affected subjects and eliminating the need for whole population studies.

Mr. Harrison inquired if the air quality has improved in the last 30 plus years why have the mortality estimates remained high. Dr. Schwartz answered that the life expectancy has increased significantly, but only in small part due to improved air quality. Even though air quality has greatly improved, the PM_{10} has had only a moderate improvement and $PM_{2.5}$ even less, as demonstrated in the Six City study. In addition, the fine particle pollution that occurs is persistent. Consequently, the levels that produce the adverse response in a susceptible portion of the population are still present. In other words, instead of seeing a large risk occasionally as was the case 30 years ago, we now see small risks every day.

Dr. Demers asked why the Seventh Day Adventist study did not show mortality effects and if the investigators collected data on this in terms of individuals with chronic bronchitis. Dr. Schwartz answered that what the investigators called the obstructive airways disease was in fact, an outcome measure that they constructed. The COPD rates in Seventh Day Adventists, are small since there are fewer susceptible individuals in it. The data that the investigators did collect, however, can be associated with air pollution. Dr. Demers asked if Dr. Schwartz would explain how the one to two years of life expectancy lost was derived. Dr. Schwartz explained that the derivation was not his and then provided a couple of explanations on how it may have been developed.

Dr. Kummler asked how weather was addressed as a confounder. Dr. Schwartz indicated that first thing that needs to be done is to recognize that the dependence on weather is likely nonlinear. On very cold and very hot days more people would be expected to die. The resulting curve would be "U" shaped. The curve used is a moderated one which adjusts for the peaks and valleys. The extreme data on either end of the curve is then ignored, thus moderating the confounder. Consequently, what is left is a narrower range of data where it may still be a "U" shaped curve, but a fairly gentle "U". In terms of the parameters, Dr. Schwartz indicated that he tends to use humidity and dew point temperature when looking at weather. Due point temperature is used because it is less correlated with temperature than is relative humidity, and it is also a measure from which a more independent effect can be obtained.

Mr. Harrison asked why wind was not taken into consideration as a factor of weather. Dr. Schwartz indicated that he did not control for wind since it was not a direct cause of mortality. Mr. Harrison stated that that was understood but the wind can in fact be the transport of the mechanism that is the cause of the mortality. Dr. Schwartz agreed that it does transmit which would suggest maybe an effect or modification strategy could be considered. He indicated that he has started looking at, preliminarily, precipitation which can also be a significant predictor of mortality in the east.

Dr. Demers questioned if it was fair to state that Dr. Schwartz's theory regarding biological plausibility was based on the fact that the fine particles are carrier systems for transition metals which may be the actual toxin, at least for cardiovascular death. Dr. Schwartz answered that transition metals were certainly a major contributor; however, there also may be other factors involved. For instance, toxicity can be produced with ultrafine carbon particles, which do not have transition metals on them. The transition metals of concern appear to be iron, nickel and vanadium.

Dr. Harkema asked if the animal studies also look at other stresses other than just the particles that may kill these animals. Dr. Schwartz answered that the only thing that has been looked at is sulfur dioxide. Investigators have not looked at ozone which probably would be a good stressor to look at since it does produce lung inflammation. Also, there are moderately consistent data on hospital admissions for lung disease and

ozone. The data on mortality have been somewhat less consistent, although more recent studies are finding associations more consistently with mortality and ozone.

Dr. Harold Humphrey (Michigan Department of Community Health) asked if the recent sophistication in asthma diagnoses skewed the findings. Dr. Schwartz replied that long term trends are considered but short term misdiagnoses are ignored since it works both ways and adds a lot of noise to the process. It was found that 90 percent of respiratory disease have been accurately diagnosed upon hospital admission.

Dr. Holcomb asked how much of the administered dose of $PM_{2.5}$ was actually reaching the lung tissues via the varied methods of dosing animals. Dr. Schwartz replied that the dosing levels were secondary to placing a dose "on" the lung tissue and observing the respiratory and cardiac response. The issue is that when a dose does reach the lung there are electrocardiogram changes.

Mr. Leonard inquired if the reduction of sulfate would result in a corresponding drop in trans-metallic and carbon $PM_{2.5}$. Dr. Schwartz responded that transitional metals appear to be carried on sulfate particles and therefore the reduction of sulfate levels should reduce airborne transitional metals. Sulfates do not appear to be entirely innocuous in themselves because of the high rate of respiratory disorder in the northeastern U.S. where they are high. Also it appears that all combustion gases have particles carrying transitional metals.

Dr. Wolff stated that the concentrations of the transition metals are probably in the ng/m^3 range which represents a very minor trace concentration of the trace amount of PM. Dr. Schwartz agreed but indicated that the hydroxyl radical formation off them is catalytic which can result in a large multiplier effect.

Dr. Wolff asked if Dr. Schwartz thought the ultrafines, rather than the $PM_{2.5}$ were the real problem. Dr. Schwartz answered that he was skeptical about that since (1) there are conflicting studies currently on this issue and (2) the ultrafine particles go into the accumulation mode in a short period of time.

Dr. Wolff stated that he was concerned with (1) some of the reanalysis studies which appear to demonstrate that if other pollutants are added, a PM signal is not necessarily the end result, (2) the nonsensical conclusions, such as ozone related mortality in the wintertime, of some of the investigations and (3) the apparent inability by others to duplicate Dr. Schwartz's investigation. Dr. Schwartz stated that in terms of the reanalysis of his study, the investigators had initially introduced a slightly different factor which changed their results. They have since corrected for this and have been able to duplicate the study. He agreed that if multiple pollutants are considered, weird results like ozone in the winter may occur; however, there was no reason to even look at ozone in the winter. The best way to deal with hypotheses about the other pollutants, is to look by restriction at places where they are correlated with particles and see if particle signal still occurs. Similarly, the best way to find out whether there is an independent association for ozone is not to do a study in Mexico City, where a high correlation would

be expected but rather to look at towns in the western U.S. where the particles are really only there in the winter.

Dr. Wolff asked about the differences (i.e., for instance, the different statistical models employed, the different lag times considered and the variation on how the meteorology is used) in the models used in the various studies and whether Dr. Schwartz used an iterative procedure to choose the final relationship. . Dr. Schwartz indicated it was an iterative procedure. Dr. Schwartz also indicated that in the initial studies, meteorology was handled relatively simplistically. As time has gone on, its application has become more sophisticated in order to account for the nonlinearities. Regarding the lag models, one of the issues in a time series study is that if yesterday's air pollution is used in a regression model, that does not represent just yesterday's air pollution, because yesterday's air pollution was correlated with the air pollution of the day before. Consequently, there is some serial correlation in the air pollution, and that varies from location to location, depending on the weather pattern and geographical location. Given this, Dr. Schwartz indicated that he was not surprised to see the variability. He noted, however, that the variability always falls within a week. The most common lag time is two days and the bulk of the studies fall between a one and three-day average.

Dr. Harkema wondered how many more epidemiological mortality studies would be necessary to make a case against PM. Dr. Schwartz indicated that in terms of time series studies, none; but in terms of cohort studies, one or two more would be useful.

Dr. Kummler questioned if standards for both $PM_{2.5}$ and PM_{10} were needed. Dr. Schwartz stated that his personal preference would be $PM_{2.5}$ only; however considering the exacerbation of asthma, a PM_{10} standard also has merit.

V. PANEL DISCUSSION

Dr. Kummler indicated that he is convinced that the studied effects have nothing to do with particles, but are the result of chemicals that are being carried by the particles. He wondered whether the question will really become one of control strategy, dealing with the precursors; i.e., controlling for various pollutants, depending on the area of the country, rather than controlling for particulate matter in general. Dr. Harkema responded that it is only an assumption at this point, and although Dr. Schwartz's hypothesis was clear, it was as yet unverified. Dr. Wolff said that there is some work being done by the USEPA, but it has not been peer reviewed yet. He also disagreed with Dr. Kummler's statement that fine particles differ vastly from one area of the country to another, saying that there are really only nitrates, sulfates, organic carbon, and elemental carbon. They will exist in varying proportions depending on the area. Dr. Kummler commented that this may create problems for USEPA's ozone strategy. Dr. Wolff indicated that for ozone, NOx and VOCs would be targeted, and for PM, NOx, VOCs, SO_2 and primary particulates would be targeted. Because the two overlap, the USEPA is indicating it may back off the ozone standard, since it will all be covered under $PM_{2.5}$.

Dr. Kummler asked the Panel if the consensus was that the health effect is caused by a particle below 2.5 microns, independent of chemical composition. Mr. Harrison said he didn't think so, since the focus appears to be on the transition metals. He suggested that maybe Drs. Fischer and Harkema should look more closely at the toxicology of the metals. Dr. Demers indicated that the common denominator for the health effects is particle size, with small particles able to enter the deepest airways and alveoli, where the damage is caused. They may either cause primary damage or serve as a vehicle onto which transition metals or other things are attached.

Dr. Wolff indicated that the USEPA's Clean Air Scientific Advisory Committee (CASAC) was convinced that the $PM_{2.5}$ is different from larger particles, and that it should be regulated separately. However, there was no agreement of the link between $PM_{2.5}$ and mortality. There are three studies that link $PM_{2.5}$ with mortality, and two of those are actually the same study, the Six City study, analyzed in different ways. The third is the Pope study. The Six City study does a direct comparison of PM_{10} and $PM_{2.5}$, but is flawed because of the large measurement error for coarse particles. Consequently, there is really no definitive direct comparison.

Dr. Holcomb requested that the documentation of the methodology and the physical history of the animals used in the studies be acquired, so a comparison in animal laboratory exposure and real world human exposure could be made. He stated that, as with CASAC, he agreed that some small particulates less than PM_{10} were health impacters but that the measure of $PM_{2.5}$ was in doubt and the real impacters are much smaller.

VI. PANEL ASSIGNMENTS

Dr. Wolff agreed to begin working on the assumptions underlying the USEPA's proposed regulations. Mr. Harrison asked Dr. Harkema to let Dr. Fischer know about the need for a toxicological analysis of the transition metals issue and the issue of biological plausibility. Mr. Harrison asked that Dr. Kummler to address the question of the lack of available and consistent monitoring data on $PM_{2.5}$ and on the potential problems with the ASTM standard methodology, which reportedly will miss some PM. Dr. Demers asked that Mr. Harrison send copies of the overheads that Dr. Wolff used regarding some of the USEPA assumptions.

VII. NEXT MEETING DATE

Mr. Harrison indicated that his office would contact the Panel members to schedule the next meeting.

VIII. ADJOURNMENT

The meeting was adjourned at 2:00 PM.

Respectfully submitted,
Keith G. Harrison, M.A., R.S., Cert. Ecol.

Executive Director
Michigan Environmental Science Board

Attachment 1. Synopsis of Dr. Joel Schwartz's March 26, 1997 Presentation to the Michigan Environmental Science Board Air Panel.

Dr. Schwartz began his presentation by discussing the evolutionary function of the human lung and its defense against bioaerosols and dust. The lung's primary defenses, the nasal passages and the mucociliary ladder, evolved to capture particles and allow the cilia to move particles out of the lung. It is a relatively effective mechanism for removing the particles that mammals were exposed to during evolutionary time periods. However, the development of fire and the subsequent new kinds of particles generated through combustion, differ in size and chemical composition from particles the lung evolved to deal with. One of the key differences is size, which determines what happens relative to the primary pulmonary defenses. The PM_{10} standard was chosen because it is roughly the demarcation between particles that get past the first pulmonary defenses and down the throat.

The larger particles, that do not come from combustion sources, deposit primarily in the upper airways, where the mucociliary ladder is located. The fine combustion particles, however, are much more likely to deposit in the pulmonary region of the lung, past the primary defenses. That is likely to have something to do with the differences in relative toxicity of the particles. But small particles differ in composition as well as deposition characteristics. Direct instillation studies done in Edinburgh and in North Carolina have found that fine particles are much more toxic than coarse particles. When particles were washed or chelated to bind off metals, toxicity was dramatically reduced. The instillation of the same amounts of coarse particles had no effect on the subjects. The solutions containing the extracted transition metals were just as toxic when instilled alone. The toxicity of the particles seems to be associated with the amount of transition metals, and transition metals are found in, and are more bioavailable from, fine particles. The fine particles release transition metals more easily and they are doing it in the pulmonary region of the lung. A National Institute of Health study found that particles collected in parts of Mexico City where combustion sources dominated were much more toxic in *in vitro* studies of lung epithelial cells than particles collected from areas dominated by dust particles. The inflammatory effects of the small particles include increased neutrophils, the lavage fluid evidence of increased lung permeability, increases in the expression of TNF alpha and MIP 2 by the neutrophils and increases in cytolytic activity as a result of these instillations.

Dr. Schwartz indicated that Dr. Dan Costa has recently reported new data on dogs with monocrotaline induced lung inflammations. The dogs were instilled with urban air particles collected from Washington, D.C. Compared with healthy control animals also instilled, the asthmatic animals showed stronger effects. In addition, inflammation increased in response to allergic triggers that were delivered with the particles. There are also inhalation, rather than instillation, studies that show that the presence of bronchitis dramatically increases the effects of small particles. John Godleski, at Harvard, exposed both bronchitic and healthy rats to particles taken from Boston air, which met permissible standards of pollution. None of the healthy rats died, but 19

percent of bronchitic rats did. They died in their sleep, and autopsies showed an increase of MIP 2 in the hearts of the rats that were exposed to particles. There were increases of TNF Alpha and MIP 2 in the lungs. Healthy dogs have been exposed to fine particles, $PM_{2.5}$, in concentrations of about 200 μg for six hours, with 24 hour averages of 70 μg to 80 μg. By the second day there were electrocardiogram changes, including "R" wave notching and alternating magnitudes of "T" waves. These kinds of changes have been established in the electrocardiogram literature as substantial risk factors for arrhythmia and for sudden death in general. That is interesting, because the largest increase in cause of death in London in 1952 was sudden death. They have also shown that if they induce a partial blockage of the coronary artery to simulate ischemia in dogs, the effects of the particles are magnified, suggesting that people who have underlying ischemic heart disease may be at greater risk of these changes and potentials of arrhythmia. Dr. Costa also has done an instillation study which produces arrhythmia with exposure to fine particles. The Edinburgh group has done studies looking at DNA plasma, showing that these particles are producing hydroxyl radicals, and that the effects can be suppressed with agents that suppress hydroxyl radical production. The fact that the effects are substantially enhanced in sick animals is consistent with the epidemiological study findings showing that people who are more prone to dying in the epidemiological studies have tended to be people with pre-existing inflammatory lung disease, COPD, or some other heart condition. It appears that PM matter exacerbates conditions with inflammatory processes, at least where acute effects are seen.

In summary, (1) fine versus coarse particles can get past the primary pulmonary defenses of the lung, (2) fine versus coarse particles have high levels of soluble bioavailable transition metals which can generate hydroxyl radicals, (3) fine versus coarse particles, can induce mortality and substantial inflammation when instilled into animals, and (4) fine particles can cause electrocardiogram disturbances and death at relevant exposures.

In terms of human studies, the greatest increases in deaths in London in 1952 were from pneumonia, bronchitis and sudden death. There was an increase in respiratory contributing causes in heart disease victims. Other cases of high air pollution are different. When Mt. St. Helens erupted, there were airborne concentrations downwind that exceeded 10,000 $\mu g/m^3$ of coarse non-combustion particles. A downwind camp was being studied, but nothing much happened to the children there. There was a study done in Montana at the same time, where concentrations were much lower, only a few thousand micrograms. Nothing much happened there either, compared with the traumatic events in London at the same level of exposure. The difference is that the London particles were from combustion, from coal burning. Episodes in Donora, Pennsylvania and the Ruhr Valley in 1985 show the same differences. The epidemiology studies are fitting in with these studies, suggesting that the toxicity to animals' lungs is in the fine mass.

Dr. Schwartz indicated that even low concentrations of PM are of concern. He looked at 3,000 days in Philadelphia; the five percent of highest air pollution days and the five

percent of lowest. There was some increase in all cause mortality, about 1.07 relative risk. Respiratory contributions on death certificates increased, as well as symptom ICD codes for respiratory symptoms. Deaths from ischemic heart disease increased during high air pollution days. "Dead on arrivals" incidents were elevated more than the overall increase in deaths. This is consistent with the London experience, but at a lower order of magnitude. It is also consistent with the animal data, which suggest that more dramatic inflammatory changes and arrhythmia occur in animals with some pre-existing inflammatory condition.

Today, in London, most particles are from diesel fuel which is used in a substantial portion of transportation. There is still a significant association with particulate air pollution and daily deaths in London; the particles are different, but still are derived from combustion. There are literally dozens of time series studies that seem to show that there are associations of particles with daily deaths, although many include other air pollutants. The time series studies all seem to show dose dependent relationships. They are found at different places with different levels of other coincident air pollutants and different weather patterns. The percent increase in PM_{10} of respiratory hospital admissions is greater than the percent increase in deaths from all causes, which is what is to be expected if the relationship between particles and respiratory cause of death is a causal relationship. The relationship holds no matter what the source of the combustion particles. The slope is not always the same, depending on conditions, but the relationship is there.

Based on data from the Harvard Six City study which show a significant increase in death rates across the range of exposures for fine particles much greater than for coarse, fine rather than coarse particles appear to be causing the problems. When coarse and fine particles are looked at separately, the epidemiology finds that coarse particles are not associated with health effects. That fits with the animal data, where there is not much effect when coarse particles are instilled in the lungs of rats. Dr. Schwartz does not think the results are affected by the greater measurement error for coarse particles, since, according to him, the error cancels itself out over many observations.

The 24 City study looked at 24 cities in the US and Canada, recruiting a random sample of about 1,000 children per community, measured their lung function, got medical histories, parental histories, etc. They found that abnormal lung function, defined as 85 percent or lower function, was significantly related to average annual fine particle concentration. These were averaged out over a long period of time. The epidemiology and animal studies together make a strong case for adverse effects from fine particles, and not coarse. The fine particles get to the deepest parts of the lungs, have toxic components, produce toxicity in animal studies, and kill animals at concentrations not much higher than those typically found in the US today.

The evidence for particles rather than sulfur dioxide being the problem come from a variety of animal and other studies which have shown that (1) unlike the fine particle studies, high exposure to sulfur dioxide did not cause mortality, (2) unlike the fine

particles studies, there were no changes in electrocardiograms in the sulfur dioxide studies, and (3) unlike in the case of fine particles, sulfur dioxide does not deposit in the pulmonary region of the lung. These studies suggest that it is much more likely that fine PM rather than sulfur dioxide, is causally related to mortality. Dr. David Fairley looked at Santa Clara, California, where there is no sulfur dioxide, but still found an association between particle pollution and daily deaths. Dr. Arden Pope has looked again at Provo, Utah, where there also is no sulfur dioxide, and has found the same thing. Dr. Schwartz indicated that he has a paper in press on Tucson, which looks at hospital admissions for heart disease. There are associations with PM_{10} and carbon monoxide, but none with sulfur dioxide.

In terms of the chronic effects and the loss of life in years, the time series studies do not shed much light on that. The Six City study was a prospective cohort of adults recruited in 1975 and followed over time. The investigators found, after controlling for individual risk factors, a significant association between fine particles concentrations and the adjusted risk of dying. They were reluctant to believe it initially, but it was confirmed by the American Cancer Society's study of 151 communities. It seemed to be a reduction of life expectancy of one to two years. Abbey's Seventh Day Adventist Study, another prospective cohort study, looked at survival rates. Initially Abbey saw no association between PM and all cause mortality. His latest data, however, after a few more years of follow-up, do show that relationship. That will be reported this year. Another long term effect shows up in the Six City study in a cohort of children. There the investigators found that chronic cough in children was related to long-term exposure to air pollution.